Oikos

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Appendix 1.

The wild boar submodel

Reproduction

Females reproduce only once a year, depending on their age class (they have to be at least subadult). Individual females reproduce depending on the season with a peak in April and no reproduction in winter from October to December (Boitani et al. 1995). Within one month the proportion of reproducing females is equally assigned to four weeks with the exception that every third month it is assigned to five weeks so that a model year sums up to 52 weeks.

Habitat quality was reflected by the maximum number of females allowed to reproduce in a herd (breeding capacity) and was calculated from field data (Fernandez et al. 2006). Older females breed first. If adult females have died due to any stochastic process, the sub-adults can also reproduce. The number of piglets per reproducing female ($N_{Piglets}$) follows a Gaussian distribution around the mean and standard deviation found in literature (Table A1).

Natal dispersal

Female groups split up once a year in summer (week 28) if carrying capacity has been exceeded in the home-range cell and not all neighboring home range cells are occupied yet by another female group. The female subadults, at least two individuals in our model, then randomly move to the next suitable cell. We allow for moves up to three cells, reflecting the reported mean dispersal distance of 6–9 km. Female groups use their home ranges exclusively, i.e. no other female group can move to that cell. Normally, female groups are very stationary, but group splitting could be an important aspect in population dynamics when female groups die out due to disease mortality. We did not consider long-distance dispersal as we only consider perfectly connected habitat and thus spatial gaps for the virus transmission due to habitat fragmentation do not occur.

Baseline mortality

Mortality is age-dependent and adjusted to annual survival estimates found in the literature. These survival estimates together with the reported variability determine the Gaussian distributions we draw from the random survival in the model on a yearly basis (SP_{Year}). The stochastic effect resembles 'good' or 'bad' years for boars, i.e. environmental noise. In the application the Gaussian distributions are cut symmetrically around the mean (Table A1). Per time step we apply the adjusted age- dependent mortality (PM_{Week}) to the individual:

$$PM_{Week} = 1 - (SP_{Year})^{1/52} \quad (1).$$

The CSF submodel (Fig. A1)

Postnatal infection

The number of infectious animals in the group I_G is counted, and with a certain effective infection probability P_{INF_G} (that combines contact and transmission) other members of the same group become infected. Additionally an animal in the group gets infected by I_N infectious neighbours. The effective infection probability P_{INF_N} is determined as a fraction f of P_{INF_G} (Table A2), since contact between animals of different cohorts is normally lower. Thus, the probability P_{SI}, that a susceptible animal becomes infected, calculates as

$$P_{SI} = 1 - \left(1 - P_{INF\_G}\right)^{I_G} * \left(1 - P_{INF\_N}\right)^{I_N} \quad \text{with} \quad P_{INF\_N} = f * P_{INF\_G} \quad (2).$$

Response to infection

After infection, the individual is not infectious for one week (incubation period) before onset of the infectious period. Mortality probability of infected individuals is dependent on the virulence of the virus, i.e. its ability to kill its host, and other individual traits of the pig, such as age and health condition. For example, when healthy pigs in good condition get infected the outcome of infection with a moderately virulent virus strain is transient or chronic. On the other hand, pigs under stress infected with a highly virulent strain become rather acutely infected, and most of them will die within a short time period. To simplify communication, we will refer to outcomes of low, moderate or high 'virulence', although we bear in mind that the outcome is not only determined by the virulence of the virus strain, but also by several factors of the host (physical condition, age; for further discussion see Gandon et al. 2002).

The virulence concept is reflected in the model as follows: First, we determine randomly whether an animal responds transiently to an infection based on the binomial parameter P_{TRANS} (i.e. infectious for one week and then having a latency period for three further weeks before acquiring an immune status; within the latency, the animals cannot be super-infected, Christoph Staubach pers. comm.). To reflect known differences in the proportion of transients within the age classes we apply

$$\begin{aligned} P_{Trans}\text{Adult} &= 1 - (1 - P_{TRANS})^2 \\ P_{Trans}\text{Subadult} &= P_{TRANS} \\ P_{Trans}\text{Piglet} &= 1 - (1 - P_{TRANS})^{0.5} \end{aligned} \quad (3)$$

If not transient, the infection runs lethal in the respective individual. For these animals we determine their individual infectious period T_S randomly from the following survival function depending on parameters maximum survival time T_{MAX} and a shaping exponent X:

$$P_{SR}(T) = \begin{cases} \left(1 - \frac{T}{T_{MAX}}\right)^{X} & \text{if } T < T_{MAX} \\ 0 & \text{else} \end{cases} \quad (4)$$

We then draw from this distribution the individual survival time T_S for each individual (Fig. 1 in the main text). Thus, disease outcome is expressed as a combination of case mortality (transient vs lethal) and the survival time of lethally infected animals. The proportion of infected pigs showing acute (living at most four weeks) or chronic outcome (living between four and T_{MAX} weeks) is determined by the exponent X, with high values of X resulting in many acute infections, and low values of X in many chronic infections, respectively (Fig. 1). These rules correspond to knowledge gained in experiments with domestic pigs when low virulent strains affected a high proportion of the animals only transiently, whereas high virulent strains killed most of the infected animals.

Prenatal infection (part of the reproduction submodel)
When the female boar is infected, 10/16 of the foeti are aborted, half of the rest consist of prenatally infected (PI) offspring, and the remaining are normal susceptible piglets (Dahle and Liess 1992). The PI piglets are automatically removed (mortality probability = 1) after time step T_S drawn from the distribution described above, i.e. PI piglets were treated like lethally infected piglets.

Partially protected piglets
If the pregnant sow is already immune, then the piglets are born with maternal antibodies. This means that, due to maternal antibodies, piglets do not become infected for the first three months (Depner et al. 2000). After that, they have a low antibody titer for TS_{MA} time steps, which makes the outcome of an infection transient. These so-called partially protected piglets are set back to the status 'susceptible' after TS_{MA} time steps unless they have been infected meanwhile and thus end up in immune state.

References

Bieber, C. and Ruf, T. 2005. Population dynamics in wild boar *sus scrofa*: ecology, elasticity of growth rate and implications for the management of pulsed resource consumers. – J. Appl. Ecol. 42: 1203–1213.

Boitani, L. et al. 1995. Demography of a wild boar (*Sus scrofa* L.) population in Tuscany, Italy. – Gibier Faune Sauvage 12: 109–132.

Dahle, J. and Liess, B. 1992. A review on classical swine fever infections in pigs: epizootiology, clinical disease, and pathology. – Compar. Immunol. Microbiol. Infectious Dis. 15: 203–211.

Depner, K. R. et al. 1997. Influence of breed-related factors on the course of classical swine fever virus infection. – Vet. Rec. 140: 506–507.

Depner, K. R. 2000. Transient classical swine fever virus infection in wild boar piglets partially protected by maternal antibodies. – Dtsch Tierarztl. Wochenschr. 107: 66–68.

Dewulf, J. et al. 2004. Analytical performance of several classical swine fever laboratory diagnostic techniques on live animals for detection of infection. – J. Virol. Meth. 119: 137–143.

Fernandez, N. et al. 2006. Viability and risk assessment in species restoration. Planning reintroductions for the wild boar, a potential disease reservoir. – Ecol. Soc. 11: 1–6.

Focardi, S. et al. 1996. The population modelling of fallow deer and wild boar in a Mediterranean ecosystem. – For. Ecol. Manage. 88: 7–14.

Gandon, S. et al. 2002. The evolution of parasite virulence, superinfection, and host resistance. – Am. Nat. 159: 658–669.

Howells, O. and Edward-Jones, E. 1997. A feasibility study of reintroducing wild boar (*Sus scrofa*) to Scotland: are existing woodlands large enough to support minimum viable populations? – Biol. Conserv. 51: 77–89.

Jezierski, W. 1977. Longevity and mortality rate in a population of wild boar. – Acta Theriol. 22: 337–348.

Kaden, V. et al. 2004. Studies on the virulence of two field isolates of the classical swine fever genotype 2.3 Rostock in wild boars of different age groups. – J. Vet. Med. B 51: 202–208.

Kaden, V. et al. 2005. Classical swine fever (CSF) in wild boar: the role of the transplacental infection in the perpetuation of CSF. – J. Vet. Med. B 52: 161.

Kern, B. et al. 1999. Incidence of classical swine fever (CSF) in wild boar in a densely populated area indicating CSF virus persistence as a mechanism for virus perpetuation. – J. Vet. Med. B 46: 63–67.

Leaper, R. et al. 1999. The feasibility of reintroducing wild boar (*Sus scrofa*) to Scotland. – Mamm. Rev. 29: 239–259.

Mengeling, W. L. and Packer, R. A. 1969. Pathogenesis of chronic hog cholera: host response. – Am. J. Vet. Res. 30: 409–417.

Narita, M. et al. 2000. Comparative immunohistopathology in pigs infected with highly virulent or less virulent strains of hog cholera virus. – Vet. Pathol. 37: 402–408.

Truvé, J. and Lemel, J. 2003. Timing and distance of natal dispersal for wild boar *Sus scrofa* in Sweden. – Wildlife Biol. 9: 51–57.

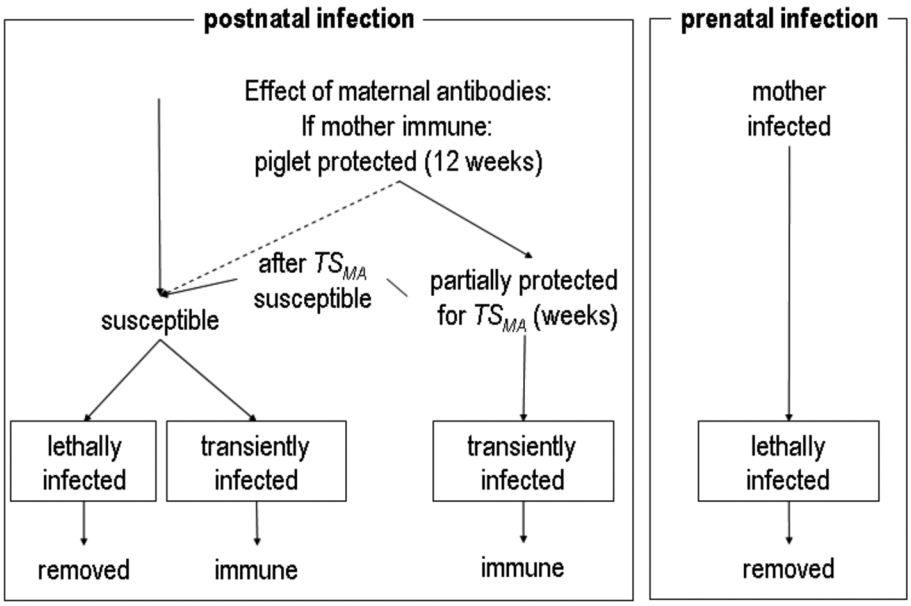

Figure A1. Flow chart of the disease course. Both lethally and transiently infected individuals shed the virus and can infect other individuals. TS_{MA} = time steps of partial protection by maternal antibodies (hypothesis 5, Table 1, see text for details).

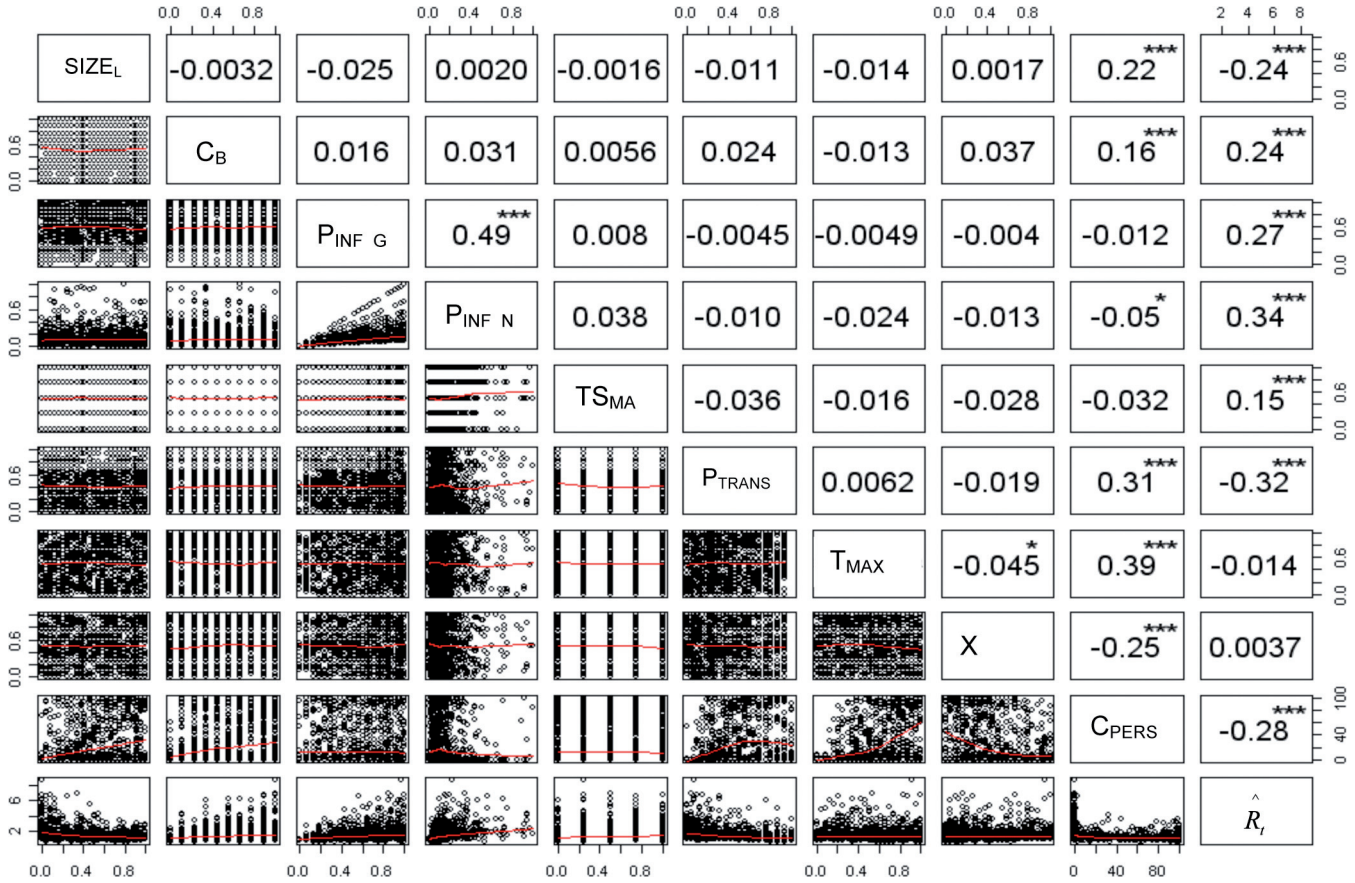

Figure A2 (A)

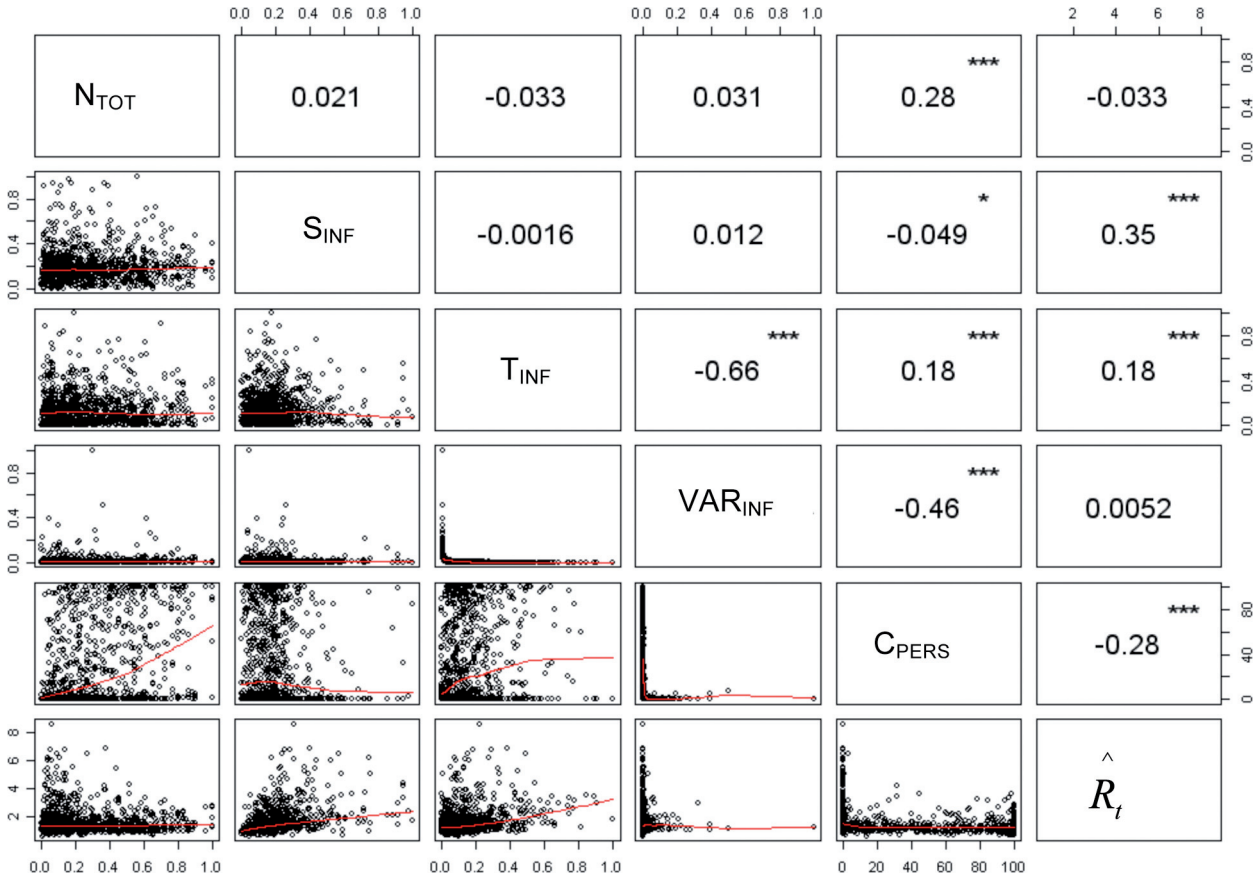

Figure A2 (B). Figure A2. Kendall's tau partial rank correlation between first-order (A) and second-order (B) independent variables and the two response variables. Asterixes show the significance level.

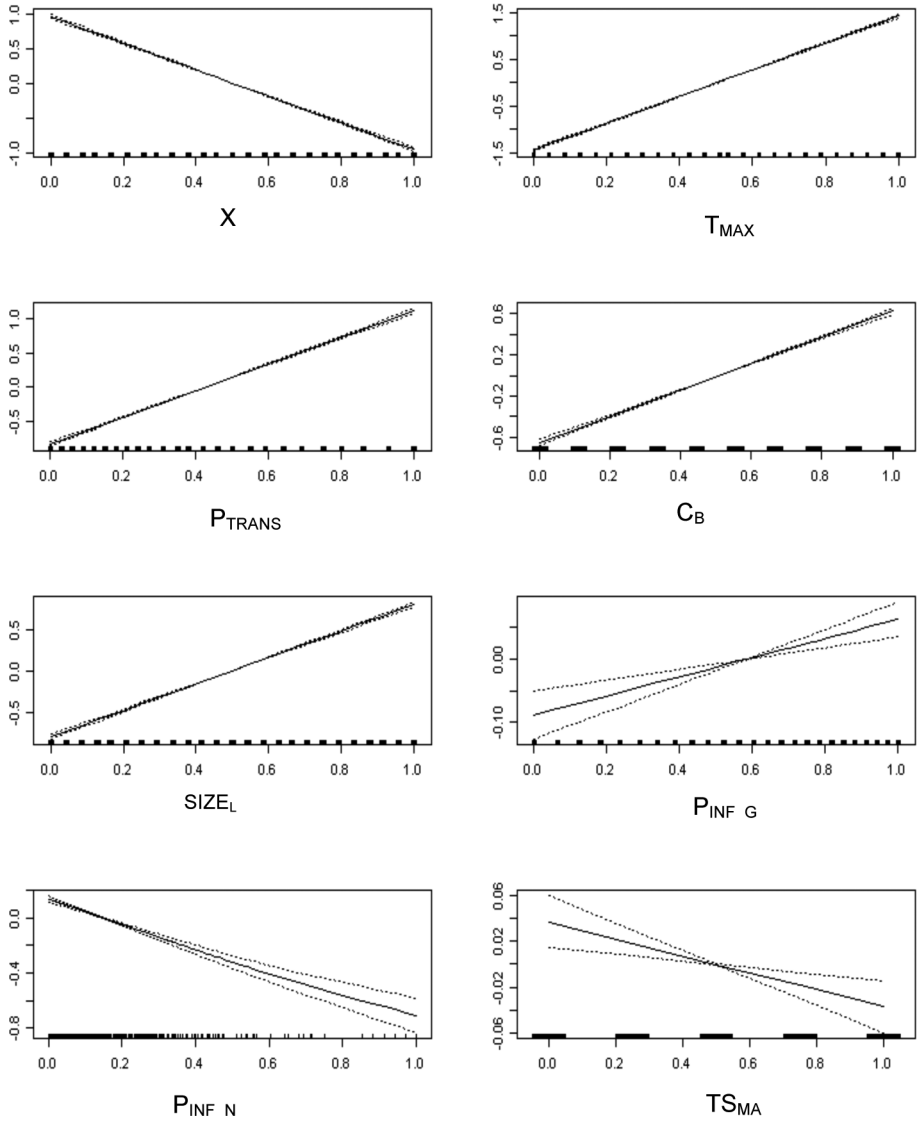

Figure A3. Structural dependency of the independent parameters for disease persistence analyzed with a GAM. -axis: partials of the respective variable.

Table A1. Wild boar population demography parameters as used in the model.

Demographic process	Symbol	Model parameter, standard deviation (sd), {range}	Reference
Maximum age (years)	Y_{max}	11	(Jezierski 1977)
Number of piglets per female	N_{piglet}	3.2, sd = 1.68, range {0 – 10}	(Focardi et al. 1996, Bieber and Ruf 2005)
Survival rate of piglets	SR_{piglet}	0.48, sd = 0.37, range {0 – 0.96}	(Focardi et al. 1996, Bieber and Ruf 2005)
Survival rate of yearlings	$SR_{yearling}$	0.6	(Bieber and Ruf 2005)
Survival rate of adults	SR_{adult}	0.64, sd = 0.24, range {0.28 – 1}	(Bieber and Ruf 2005)
Natal dispersal distances of sub-adult females	D_{natal}	up to 9 km	(Truvé and Lemel 2003)

Table A2. Model parameters (1 and 2), their ranges and response variables (3). Parameter combinations were assigned by Latin Hypercube sampling out of 25 equal intervals for each range. The intervals of P_{INF_G} and P_{TRANS} are determined on a logarithmic scale. Parameter values for scenarios testing hypothesis 3 [H3] are given in parentheses. Second-order parameters are calculated from the parameter values of the first order parameters.

(1) First-order parameter description	symbol	Parameter value [H3]	References, e.g.
Effective infection probability within herd	P_{INF_G}	0.005 – 0.85 [0.05, 0.1]	–
Effective infection probability between herd being the fraction of P_{inf_G}	P_{INF_N}	(0.1–1)* P_{INF_G} [0.1,0.5]	–
Number of weeks, where piglet/ subadult is protected by maternal antibodies	TS_{MA}	12 – 16 [12, 14]	(Depner et al. 2000)
Landscape size (number of cells). One dimension is standardized with 25 cells	$Size_L$	10 – 150 [100]	–
Breeding capacity per home range (cell)	C_B	1 – 10 [5]	(Leaper et al. 1999; Howells and Edward-Jones 1997)
Presence of PI piglets	PI	Yes, no [H3]	(Kern et al. 1999; Kaden et al. 2005)
Maximum survival time (weeks) of lethally infected boars*	T_{MAX}	5 – 52 [5, 30]	(Depner et al. 1997; Mengeling and Packer 1969)
Exponent, giving the proportion of chronic and acute infections **	X	1 – 10 [1, 3]	(Narita et al. 2000; Kaden et al. 2004)
Probability of transient infection (for subadults; values for adults and piglets have to be calculated with the formula described in the Appendix)	P_{TRANS}	0 – 1 [0.2, 0.8]	(Dewulf et al. 2004)
(2) Second-order parameter description			
Total population size	N_{TOT}		= C_B * $SIZE_L$ * 25
Mean infectious time of an infected individual	T_{INF}		Depending on P_{TRANS}, T_{MAX} and X (see eqn. DA-4)
Mean-to-variance-ratio	VAR_{INF}		T_{INF} / variance (see eqn. DA-4)
Overall infection probability defining the speed of spread	S_{INF}		= (P_{INF_G} + (8 * P_{INF_N})) / 9
(3) Response variable description			
Counts of disease persistence events in 120 repetitions	C_{PERS}		count
Mean number of new infections per infected individual	$\hat{R}_t$		Mean(Newly infected I_{NEW} / already infected I_{OLD} individuals per time step) * T_{INF}

* Note that when T_{MAX} = 5 we have only acute infections *per definitionem*.
**E.g T_{MAX} = 15 and X = 1 refers to a proportion of 47% acute vs. 53% chronic infections, X = 3: 73% acute vs. 27% chronic infections, X = 10: 97% acute vs. 3% chronic infections. (Note that only lethal infections, i.e. non-transient infections, cause chronic disease).